THE RETICULAR FORMATION REVISITED

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The reticular formation (RF) is one of the most fruitful conceptions of recent years in the field of the physiology and pathology of the central nervous system. Yet many aspects of the anatomy and physiology of the RF are still shrouded in the haze of uncertainty and doubt, owing partly to technical difficulties and partly to the difficulty of correlating the findings obtained in histological and in physiological investigations. Thus the term "RF" has come to mean different things to the anatomist, the histologist and the physiologist.

For the anatomist, the *formatio reticularis*, or white and grey reticular formation, is a morphological unit consisting of nervous tissue filling the interspaces among the large ascending and descending fibre tracts and sensory and motor nuclei of the brain-stem.

For the histologist, the RF is not a morphological unit but an aggregate of different units or nuclei, giving origin to nerve fibres with different destinations, lengths and numbers of collateral branches. Recently the number of nuclei in the RF of the cat and man have been mapped out: their number runs into several scores, but to only a few of them is it possible to attribute a definite function.

For the physiologist, the RF has been for a long time a notable region containing vital centres, such as the respiratory, cardiac, vasomotor and vomiting centres. Recent investigations suggest that, in addition to these centres, the RF contains nuclei which control the action of the spinal anterior horn cells and of the cerebral cortex. Three main physiological systems may be distinguished in the RF:

1. *The reticular cerebellar system.* According to the findings of Brodal (1957) there are at least three main reticular nuclei transmitting impulses to all regions of the cerebellar cortex, and perhaps also to the deep cerebellar nuclei. At the same time the reticular nuclei are obviously key structures for the efferent cerebellar impulses, since there is no direct pathway between the cerebellum and the spinal cord. From all the deep cerebellar nuclei fibres descend to the RF and end mainly contralaterally at all levels of the dorsal tegmentum, the pons and the medulla. The diffuse endings of the cerebellofugal fibres suggest that considerable overlapping and interplay of impulses from the cerebellum and other centres may occur in the RF.

2. *The reticular descending system.*

The motor and premotor areas (areas 4 and 6), as well as the parietal and temporal cortex and the cortex of the medial side of the hemispheres, give origin to fibres which descend, in part at least, in the pyramidal tract and end in the pons and medulla. It is interesting that these pontine and medullary regions are also those from which reticulo-spinal fibres arise, and that the medullary nuclei, in which the descending cortical fibres end, are also those with neurones projecting back to the cortex. Though it is difficult to be dogmatic about this, these nuclei of the RF may act as

relay centres for transmission of impulses of cortical origin to the spinal cord and at the same time as feed-back centres of these impulses to the cortex. Fibres from the *globus pallidus, putamen,* mammillary bodies, and lateral hypothalamus end in the RF of the midbrain tegmentum. The reticulo-spinal fibres descend in the ventro-lateral funiculus, but they do not appear to reach so far caudally as the lumbar and sacral segments of the spinal cord. It may be that their impulses are relayed indirectly to the more caudal levels of the cord.

3. The reticular ascending system.

There are direct spino-reticular fibres ascending in the ventro-lateral funiculus of the spinal cord from all spinal levels. Further collateral fibres from the main sensory tracts have been demonstrated to end in the RF. Although these ascending fibres end practically at all levels of the brain-stem RF, there are two main stations in the caudal medulla and in the pons—where spinal fibres end and from which reticulo-diencephalic fibres originate. The reticulo-diencephalic fibres ascend in the RF up to the caudal border of the diencephalon and then form two divisions directed respectively to the intralaminar nuclei of the thalamus and the subthalamus. Furthermore, some fibres have been seen connecting the midbrain RF to the mammillary bodies, the lateral hypothalamus and the basal ganglia.

It should be stressed, however, that these three main systems are not self-contained and independent of each other, and this is for three main reasons. First, because of the presence of internuncial cells with short axons, believed to form a diffuse system of internal communication within the RF.Perhaps too much has been made of this system, and there are reports that the number of such internuncial neurones in the RF is smaller than expected. Secondly, the interdependence of the three main systems outlined above is brought about by the rich collateral arborization of the long axons of the reticular neurones as they ascend or descend in the brain-stem. The impulses conducted by these axons are thus capable of influencing neurones situated at different levels of the brain-stem. In fact the dendritic tree of the reticular neurones is said to be oriented horizontally, in planes perpendicular to the main axis of the brain-stem, spreading over wide areas of the region, and even inside the sensory nuclei of the cranial nerves and sensory ascending tracts, such as the medial lemniscus, the trapezoid body and the nucleus of the descending tract of the trigeminal nerve. On these dendritic trees will impinge not only the impulses ascending along the classical sensory pathways, but also those transmitted to the higher or lower centres by the RF itself. Thirdly, the neurones of the RF cannot be simply classified as ascending or descending neurones, because the axons of some of them branch near the cell body, one branch taking an ascending and the other branch a descending direction. These are the main reasons which make it difficult to consider the three RF systems as working in isolation. Herein lies the puzzle and the challenge of the mechanisms of the RF, in that it seems to be designed to work as an integrating system in which events occurring in one part of it are modulated, modified and elaborated by events in other parts of the RF and in other central organs.

Physiology of the RF

It has always been a tenet of classical physiology that the brain-stem of lower mammals must contain centres capable of producing abnormal muscle tone, rigidity or spasticity, when released from the influence of cortical centres. Through its descending system the RF exerts a powerful effect on spinal motoneurones and its action has several features worthy of note.

The RF may be divided into two parts, each having an opposite effect on skeletal muscles: a small, well-defined medial area

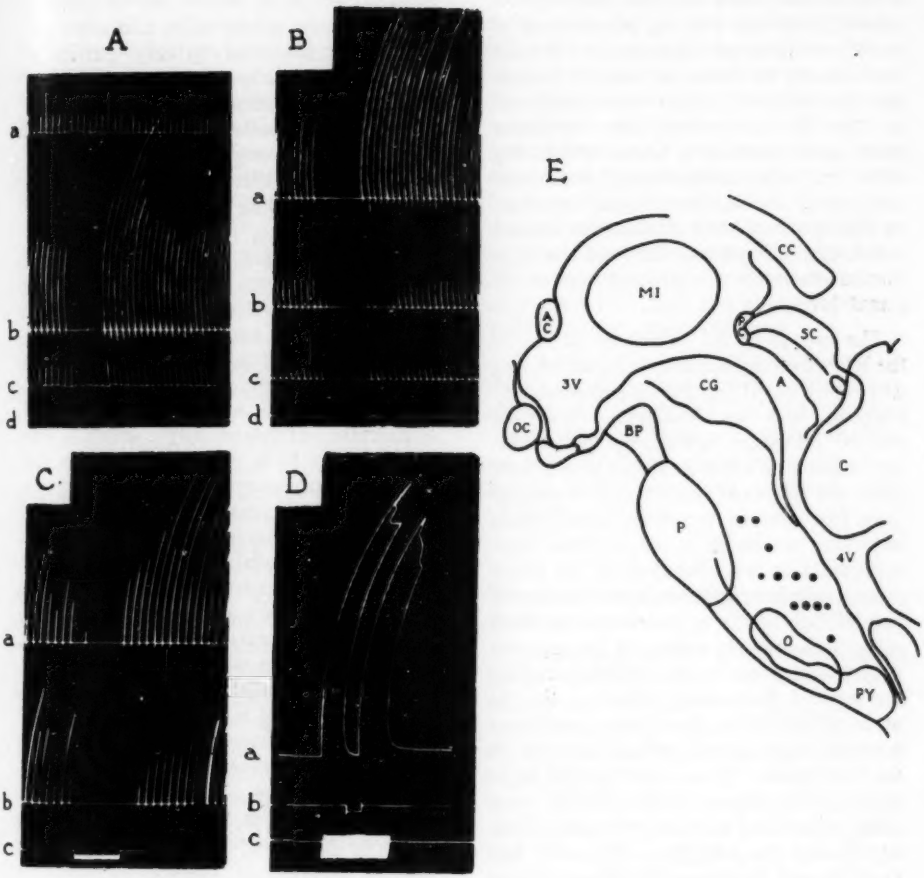

Fig. 1.—Inhibitory effects on reflex contractions (A and B) and on cortically induced movements (C and D) from stimulation of the inhibitory RF, in cats under chloralose anaesthesia. A and B: *a*, flexor reflex; *b*, patellar reflex; *c*, blinking reflex; *d*, signal of reticular stimulation. C: *a*, contraction of flexor muscle of foreleg; *b*, contraction of flexor muscle of hindleg; *c*, signal of reticular stimulation. D: *a*, contraction of flexor muscle of hindleg caused by stimulation of internal capsule; *b*, signal of reticular stimulation; *c*, signal of stimulation of internal capsule. E: points from which inhibitory responses have been obtained. (From Magoun & Rhines, 1947.)

in the bulbar RF, with an inhibitory effect on spinal motoneurones; and a larger, diffuse area extending from the caudal diencephalon to the medulla, in the lateral part of the RF, with a potentiating effect on spinal motoneurones.

1. *The inhibitory RF.*—When the function of the inhibitory RF is tested by the admittedly coarse method of electrical stimulation at 100-300 stimuli per sec., a most remarkable effect is obtained, in that a number of spinal reflexes, at all levels

of the spinal cord, may be inhibited bilaterally, and any existing muscle tone is caused to disappear (Magoun and Rhines 1947). It can be shown by suitable lesions that this inhibitory effect is not mediated by the cerebral cortex, the vestibular nuclei and descending fibres originating elsewhere, but coursing through the stimulated area. The inhibition must therefore be due to stimulation of neurones located in the medial inhibitory RF, and the location of the inhibitory process must be at spinal level.

The aspect of the inhibitory action of the RF which appeared to be most striking and unexpected in the experiments by Magoun and his co-workers was the general inhibitory action, the indiscriminate depressant effect on spinal levels. Such effect did not even respect time-honoured laws like that of reciprocal innervation, according to which it might have been expected that an inhibition of the flexor motoneurones might be accompanied by potentiation of the extensor anterior horn cells. This orderly action of the nervous system appeared to be overwhelmed by the general depressant action of the inhibitory RF. It has never been established whether these general effects are due to the inadequacy of the experimental technique, which makes it impossible to bring about a localized and discriminating effect, or whether the inhibitory RF really acts as a general brake on all spinal actions. In support of the idea of a more discreet action of the RF are some results showing that differential actions on different extensor and flexor motoneurone pools and on different types of spinal reflexes may be obtained. It is possible therefore that in the normally behaving and acting animal the inhibitory RF may exercise a subtle, discriminating and variable action on the spinal levels and act as a co-ordinating mechanism of muscular action.

2. *The facilitatory RF.*—The lateral RF, on the other hand, has a potentiating effect on spinal motoneurones. The area from which such effect can be elicited experimentally is very wide and does not seem to correspond to any particular anatomical or nuclear subdivision. In fact no long descending pathways can be tiating effect is mediated by chains of short demonstrated as originating from this area and it is likely therefore that the potenneurones descending in relays to the spinal cord.

A deeper insight into the workings of the inhibitory and facilitatory RF has derived from electrical records of the activity of single neurones of the RF obtained with appropriate microelectrodes. These studies have shown that the neurones of the RF are continuously and apparently spontaneously active, the pattern of activity being different in the different neurones examined and seemingly independent of sensory input for its inception. In some cases it has been possible to correlate the activity of single units of the RF with concurrent changes of muscle tone: for instance a sudden drop of muscle tone in a decerebrate preparation has been recorded together with an increase in the frequency of discharge of the units of the inhibitory RF. It is tempting to think of the two events as cause and effect, although this has not been shown perhaps beyond reasonable doubt. These records from single units of the RF have also shown that one and the same reticular neurone may be under the influence of a number of factors: changes in the activity of a unit have been obtained by stimulation of sensory peripheral fibres, by stimulation of the anterior lobe of the cerebellum and of the motor cortex. Furthermore, changes of muscle tension have accompanied some of the modifications of reticular activity. This convergence of impulses from such diverse nervous organs on a single reticular neurone may appear bewildering if the working of the nervous system is thought of in terms of the main classical pathways; on the other hand, nothing but such a convergence on a central integrating organ can explain the co-ordination of muscular action in the normal subject.

Experimental lesions of the RF cannot be used very successfully owing to the technical difficulties involved in making lesions in this region of the nervous system. It has been shown however that lesions of the facilitatory RF decrease the rigidity produced experimentally in animals by ablation of the motor area and of the anterior lobe of the cerebellum.

A great advance in the knowledge of the mechanism of action of the RF and the cerebellum has been made by Granit and his co-workers (Granit 1955). They have shown that supraspinal structures, like the RF, are linked to the spinal gamma motoneurones by fast descending fibres, with a speed of conduction which compares favourably with that of the largest pyramidal fibres. The action of the RF and the cerebellum is carried out mainly, but perhaps not exclusively, by controlling the rate of discharge of the gamma fibres and the resulting contraction of the intrafusal fibres of the spindle. In this way the afferent discharge from the annulo-spiral endings can be regulated, these receptor organs giving an integrated response under the combined impact of factors like the length of the intrafusal fibres, the degree of stretch imposed on the muscles, and the degree of shortening of the muscle during contraction. The supraspinal centres seem to have a double action: they alter the length of the intrafusal fibres, thus keeping the spindle afferent mechanism in the range of maximum sensitivity in all possible mechanical situations occurring during muscular action, and, by altering the spindle afferent discharge, they change at the same time the reflex excitation acting on ventral horn cells. In this way the supraspinal structures integrate motor and sensory performance in a fascinating way, achieving movement through the gamma loop and at the same time an adequate volume of sensory information from the contracting muscles.

Experimentally abnormal muscular rigidity may be obtained presumably by releasing the RF from the tonic action of the higher centres. Experimental spasticity may be of two different kinds: either the gamma rigidity, in which, following a lesion in the central nervous system, the rate of discharge of the gamma fibres is increased, with consequent reflex activation of alpha anterior horn cells and muscular tonic contraction; or the alpha rigidity, due to potentiation of the discharge of the alpha anterior horn cells by impulses from the supraspinal structures. The latter type of rigidity does not demand any reflex activation and in fact is present even in the deafferented limb.

It would be interesting to know how far these ideas derived from experimental studies can be applied to disturbances of muscle tone in human subjects: for this we need to know many more details of central and peripheral anatomy and physiology in man.

The Reticular Formation and the Cerebral Cortex

The influence of the RF on the cerebral cortex was discovered by Moruzzi and Magoun (1949) when they found that the high-voltage slow waves of the EEG of the lightly anaesthetized animal disappear following stimulation of the RF. This effect may be obtained by stimulation of wide areas of the RF in the bulbar, pontine or mesencephalic regions, and the block of the slow waves can be recorded in wide areas of the cortex, bilaterally, being more marked in the frontal region and progressively less in the parietal, temporal and occipital regions. On the other hand, in the hippocampal region the slow waves become more marked during stimulation of the RF.

It can be shown that this effect is not due to antidromic impulses reaching the cortex, or to stimulation of motor fibres descending to the spinal cord, or of sensory fibres in the spino-thalamic and lemniscal pathways. The disappearance of the slow waves of the EEG is caused by impulses ascending from the RF along

ill-defined pathways in the paramedian region of the brain-stem.

What is the functional significance of the disappearance of the slow large waves from the EEG record? The interpretation given by Moruzzi and Magoun is based on the early observation by Berger that the EEG of man at rest or asleep is characterized by slow high-voltage rhythms, and that these rhythms disappear and are replaced by fast low-voltage waves when mental activity begins or the subject is stimulated by sensory activation. At rest or during sleep large numbers of cortical neurones tend to beat together in synchrony, while during activity their rhythms become asynchronous. Generalizing from this, rest and sleep have been equated with large and slow EEG waves, and activity, arousal and alertness with fast EEG rhythms. It is to be regretted that these terms have become equivalent in the literature, although some of them have an exclusively behavioural and others only an electrical connotation. No absolute or causal relationship between these two classes of phenomena has ever been established.

The central rôle of the RF in maintaining the active rhythms of the EEG and alertness of behaviour is demonstrated also by the fact that, following lesions of the

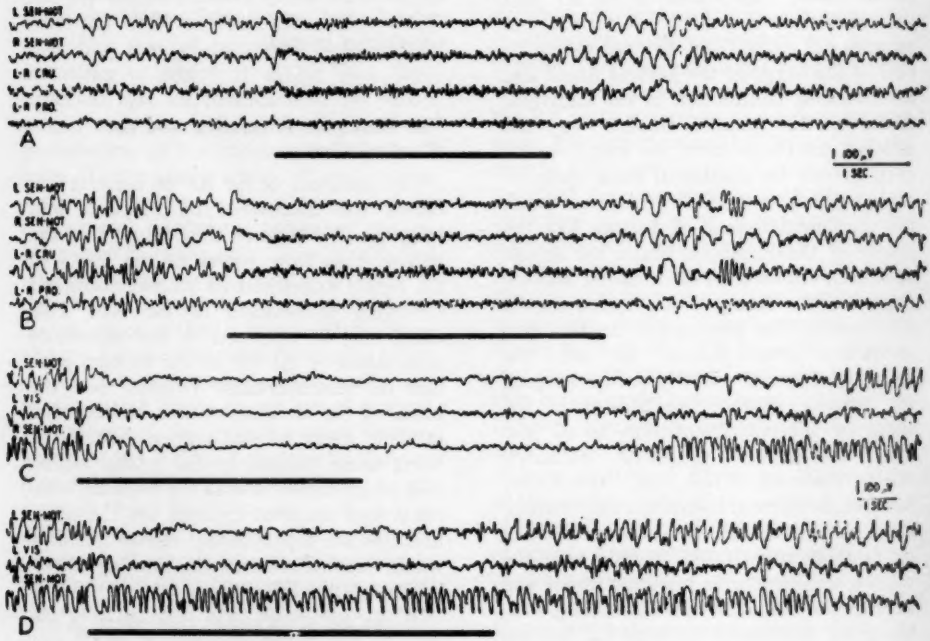

Fig. 2.—Effects of stimulation of the brain-stem reticular formation on the electrical activity of the cortex in cats under chloralose anaesthesia. A and B: Encéphale isolé preparations, 7 mg./kg. chloralose. At signal stimulate the reticular formation, 1·5 v., 300 stimuli/sec. C and D: intact cats under chloralose. At signal stimulate the reticular formation with 300 stimuli/sec. in C and 100/sec. in D. L (R) SEN—MOT=left (right) sensory-motor cortex. L—R CRU=left to right cruciate gyrus. L—R PRO=left to right gyrus proreus. L VIS=left visual area. (From *EEG Clin. Neurophysiol.* 1949, **1**, 455).

RF, normal alert behaviour and the arousing action of sensory peripheral stimulation are abolished. Cats with large lesions of the RF, but with intact spino-thalamic and lemniscal pathways, remain comatose and completely inert and can be kept alive only with considerable nursing skill. The parallel with human cases of lesions in the brain-stem is striking. Furthermore, animals with such lesions cannot be aroused, or emerge only for a short time, from their comatose state. Since in these preparations the lemniscal pathways are still intact, peripheral stimuli can reach the lateral thalamus and the cortex, but they are quite ineffective in the absence of the normal RF. On the other hand, if the spino-thalamic and lemniscal pathways are damaged, but the RF is intact, the animal behaves normally and the electrical rhythms of the cortex are the normal rhythms of alertness.

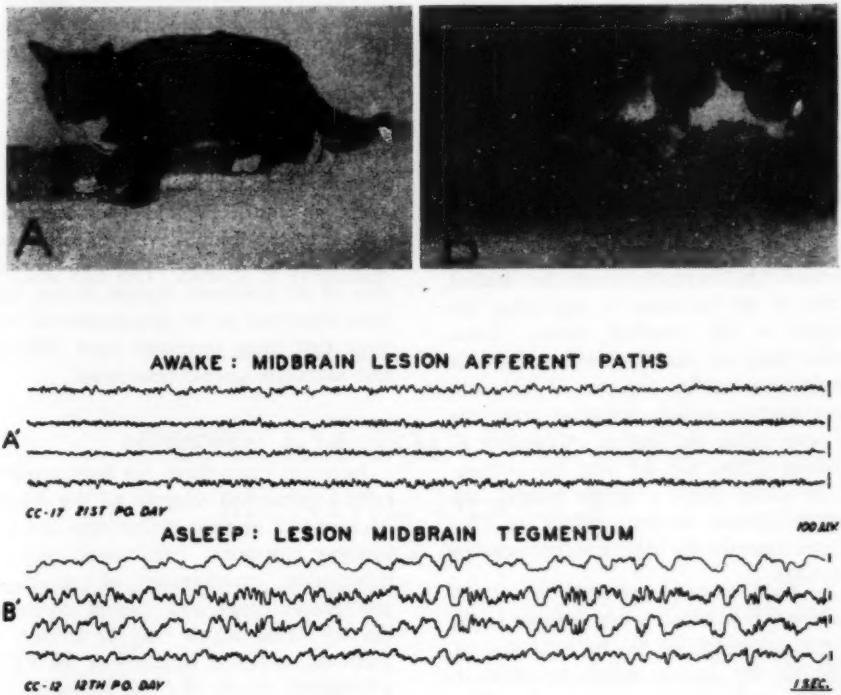

Fig. 3.—Animal with interruption of sensory paths in the midbrain, sparing central tegmentum, standing awake (A), with strip of characteristic EEG record of alertness (A'). Animal with interruption of mesencephalic tegmentum, sparing sensory paths, lying asleep, (B), with characteristic EEG record of sleep or coma (B'). (From *EEG Clin. Neurophysiol.* 1950, **2**, 483).

The same general line of evidence is valid also for "arousal" brought about by cortical stimulation. In a cortex showing slow large EEG waves, electrical stimuli can evoke rhythms of alertness only if the RF is normal. Lesions of the RF abolish

the capacity of cortical electrical stimuli to alter the electrical manifestations of cortical activity. It is thought therefore that electrical stimulation of the cortex can alter its electrical activity only through the intermediary of the RF, but it is incapable of doing so by a direct intracortical effect.

These experiments show that the normal working of the cerebral cortex and possibly alert behaviour are dependent on the normal function of subcortical centres. This general view is in keeping with the theories put forward in the 1930's by Hess, who discovered the hypothalamic sleep centre, and by Morison and Dempsey (1943). The last-named workers have stressed the importance of the medial nuclei of the thalamus in regulating the activity of the cerebral cortex. These medial thalamic nuclei, or diffuse aspecific intralaminar nuclei, are one of the pathways through which impulses from the RF can affect the cortex. According to Penfield (1954) the RF and the intralaminar nuclei form a single system, the centrencephalic system, which is considered essential for all the higher nervous functions.

If the RF has such a key position in the general economy of the nervous system, what are the factors which maintain its function and keep it—so to speak—idling along? While it is possible that in such a complex structure the activity may be maintained indefinitely without external prodding, there are several possible sources of stimulation. First, the sensory impulses reaching the RF from peripheral receptors, as already explained; secondly, the impulses from the cerebral cortex in cortico-reticular fibres; and thirdly, the levels of adrenaline, carbon dioxide and oxygen in the blood. These chemical factors are capable of influencing the electrical rhythms of the cortex by acting on it indirectly, through the RF. But if the lack of oxygen and the excess of carbon dioxide alter the cortical activity only when their blood-levels are considerably different from the normal, the sensitivity of the RF to the blood-level of adrenaline is quite considerable. Perhaps a wide generalization may be risked and we might consider the posterior and basal regions of the brain—i.e., the brain-stem and the hypothalamus—as regions containing a number of chemoreceptors or groups of nervous units specifically sensitive to certain chemical factors: chemoreceptors sensitive to carbon dioxide, apomorphine and adrenaline are known already in the RF, and chemoreceptors signalling the blood-levels of oestrogen, thyroxine, water and salts, and perhaps also of an unknown hunger factor, have been described in the hypothalamus. It is clear that these receptors must influence and modulate animal behaviour.

CONCLUSIONS

In recent years there has been a regrettable tendency to relegate all the difficult questions of nervous physiology—such as learning, memory, conditioned reflexes, habituation, mechanism of action of anaesthetic drugs and mental states—to the RF. This, which may be termed the waste-paper-basket function of the RF, is a tendency which must be opposed. The experimental evidence indicating that the RF is concerned in the regulation of muscle tone and of cortical activity is however overwhelming. But even these aspects of the function of the RF suffer from large gaps which must be filled before the experimental ideas can be applied to the human material.

The most pressing and elusive general question still unsolved is whether the RF acts as a non-specific activator of the cortex and an over-all controller of muscle tone, or whether by a continuously variable action it co-ordinates cortical action

and postural and phasic muscular movements in all physiological situations. In other words, does the RF make a difference only between coma and alertness and between spasticity and atonia, or does it also mediate all the infinite intermediate degrees between these opposite poles?

The histological investigations being done on normal and pathological human material are bound to yield useful results in demonstrating the type of organization and relations between the various systems represented in the RF, and in illustrating features which make the RF a unique and fascinating physiological mechanism.

Acknowledgements: Fig. 1 is taken from the book by Magoun and Rhines by courtesy of the publishers, Charles C. Thomas, of Springfield, Illinois; and Figs. 2 and 3 from papers in *E.E.G.* and *Clinical Neurophysiology*, by kind permission of the Editors.

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PROBLEM SOLVING AND MENTAL DEFECT

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Conditions of Mental Growth

The psychological study of backwardness has always involved two trends. The first of these, represented by Binet, has been inclined to present psychological abilities or human behaviour as if it were largely determined by innate qualities. The second point of view, represented by Binet's compatriots of the 18th and 19th centuries, notably Itard and Seguin, has always put emphasis on education. Despite the relative sophistication of modern viewpoints the dichotomy between these two attitudes has resulted in the continued belief that the backward are ineducable. This is clearly an oversimplification and in the last ten years many research studies have shown that the motor performance of the feeble-minded and the severely subnormal can be greatly improved by constant training. The work of Inhelder and Piaget in Geneva and Luria in Moscow has also encouraged us to believe that the education of young children can be considerably assisted if we understand their stages of development.

Since the second world war psychologists and pædiatricians have begun to understand that the full growth of an adult intellect is dependant not only on a sound neurological foundation but also on adequate conditions of growth. It is clear also that the primacy of general ability is not complete and that, especially in motor abilities, skill may be great in one and little in another for the same person. Likewise, in some cases high

verbal ability is found with poor motor coordination. The recognition of the need for proper conditions for full development, together with the re-examination of the concepts of the unitary nature of ability, has led to some exciting research both concerning the types of mental operation involved in learning and the factors affecting the development of each of these. It has been recognised, for example, that different kinds of deprivation have different effects. Sir Cyril Burt (1937) first drew attention to the differential effects of backwardness on operations which contribute to thought processes. Of the many factors now recognised to be involved in thinking and learning—for example, conditioning, trial and error, imitation, imagination and judgment—he singled out reasoning, memory and attention as being most frequently absent in the backward and in the order quoted here. But it is also clear that one might make other subdivisions of learning processes and test the presence or absence of such sub-abilities among mental defectives. Until 1947 there had been very few attempts to break down learning in this way, largely because of the effect of Binet's researches and the incorrect inference people drew from them —namely, that those of low intelligence were in some general sense ineducable.

In mental deficiency the possibility of multiple damage might differentially effect any one of the processes mentioned above. Psychological experiments with children and with animals in Hebb's laboratory and in Thompson's studies in Canada, as well as the studies of ethologists such as Tinbergen, have amply demonstrated the effects of some forms of deprivation on curiosity. Clarke and Clarke's (1954) work with the feeble-minded has shown that cruelty adversely affects their intelligence. Many studies in the past have indicated the serious results of poverty and malnutrition on scholastic achievement, and notable among these are studies by Binet (1908) and Allardyce (1939). In Russia in recent years Liublinskaia has demonstrated that steps in a learning process which have not been adequately learned can result in a failure of all subsequent learning steps which rest on this step in the particular subject concerned. For example, the failure to acquire and internalise the process of counting, or the failure to appreciate some aspect of numbering, can retard students in their understanding of more complex processes such as multiplication and division. In the same way vocabulary deficiencies can have a widespread effect on retarding learning in other fields. Social factors, motivation and health also play a part. As Sir Cyril Burt has said, "The health of the mother, the wages of the father, the conditions of housing, and the security and insecurity of the family as a whole, their daily experiences, the papers and books they read—these and other factors of the child's home life are bound to affect directly or indirectly the success of the work in the classroom and will largely decide its success or failure". More recently a good deal of stress has been placed on one aspect of deprivation in relation to children and it is as well to realise that the word "deprive" has many meanings. Each form of deprivation may result in reducing in a child the level of his or her efficiency not only in one particular skill but in a number of associated skills, whether these are social or scholastic.

Among the most important forms of deprivation are those that affect speech, because through speech concept formation can proceed most rapidly and the solution of problems can be greatly simplified. In a recent case of a man who regained his sight after being blind for nearly 50 years it was clear that he still relied primarily on the sense of touch for his meaningful experience, translating what sights he could see into the language of touch before giving them verbal expression. In some ways this example is an anologue of the experience of defectives, who lack the language not only to express their thoughts but even to

create them. The importance of deficiencies in thinking can in many cases be traced to verbal deficiencies, and it is in this area that studies concerning the problem solving of defectives is likely to prove most valuable. By experiments concerned with topics of this kind my colleagues and I have attempted to study learning processes, keeping in mind our belief that these are likely to prove extremely complex and that we can possibly sub-divide them, training the particular skills in which defectives may be especially backward.

Recent Work on Training Word Learning

Since 1947 a number of researchers in America and this country have turned their attention to the relationship between the level at which subjects commence their performance of a particular task and the level they finally achieve. Secondly, they have concerned themselves with the recognition of signs by mental defectives, and thirdly, they have thought about the ability of defective patients to transfer a skill gained in one task to like skills. The results of studies of the first kind show that defectives begin a task at an artificially low level compared with their final achievement. This starting level is closely related to their performance on intelligence tests but their final level is poorly related to their I.Q. This kind of finding has been made recently by McCulloch et al. (1955). The second kind of study has usually shown very considerable overlap between those belonging to different intelligence groups, and the third kind of study has shown that defectives can in fact make use of even abstract skills once these have been acquired. For example, Barnett and Cantor (1957) have demonstrated that defectives trained to discriminate between inverted and upright triangles can readily transfer this skill to discriminating between inverted and upright semicircles. Groups trained on triangles do much better with semicircles than groups which have not been so trained. In Russia Luria has also shown that many defectives have great difficulty in using words to direct their thoughts and their behaviour. He has demonstrated that children use words in at least three ways—to initiate action, to inhibit action, and as a form of self-direction. Defectives of a certain degree of backwardness have difficulty in using words to inhibit action and to direct their own behaviour.

With findings such as those reported above in mind, and considering the possibility that many severely subnormal patients had failed to acquire some essential scholastic skills, my colleague, Dr. Hermelin, recently conducted several experiments which have demonstrated in a novel way the capacities of the severely subnormal to perform mental operations of an abstract kind. The first experiment was concerned with the solution of problems by making use of learned concepts. The experimental situation involved the correct choice of one of a pair of picture cards under which a reward was hidden. In some cases the reward could be correctly obtained only by remembering a randomly determined series of pictures. In a second situation the correct card could always be picked by remembering that it was signalised by a picture of a particular kind—for example, a picture of a piece of furniture. In a third situation the reward could be obtained by remembering that the picture card concealing it always depicted several objects, and the unrewarded card depicted only one. The experiment demonstrated beyond doubt that the severely subnormal of average I.Q. 35 could none-the-less make use of concepts in the solution of problems of this kind. Where a concept was made use of about 11 trials were required to completely learn a series of cards correctly, whereas to learn a similar series without the aid of a concept required about 18 trials.

Having demonstrated that defectives of this level of intelligence could use concepts, another investigation was commenced to establish whether or not a similar group of

patients could transfer such a skill once it had been acquired. This was done by using a technique well known in psychology for measuring transposition. The relationship between two squares is learnt, for example, by always rewarding the selection of the larger of the two; the smaller of the two is then presented in a new situation together with an even smaller square. The correct solution in this instance is for the subject to select the larger of the two squares in the new situation, even though this was formerly the wrong one. On a task of this kind it transpired that defectives do almost as well as normals in learning the original discrimination and do equally well in the second test problem of transfer.

Another experiment was now carried out in which, having taught a group of subjects to discriminate between a large square and a smaller one, the situation was now reversed. Where the large one had been the rewarded choice the small one now became rewarded. In this situation defectives reversed more readily. The defectives and normal children of the same mental age learnt at the same rate, but the normal children took nearly as long to learn the reverse situation as they had taken to learn the original situation. The backward children, however, reversed much more readily. Dr. Hermelin observed that whereas the normal children could describe their mental processes in words, the backward children could not. A further stage of the experiment was planned therefore to discover if the imbeciles could reverse more readily because of being unimpeded by verbal self-instruction. By teaching a new group the same task but with added verbal reinforcement it was found that their capacity to reverse the originally learnt situation could be impaired. A conclusion was drawn from this finding that defectives generally make less use of words in problem solving than do normal children of the same mental age.

This conclusion was in keeping with similar findings of Professor Luria, who listed several functions of language as found in young children. The impelling power of language was the first, its use to inhibit an action the second, and its function of directing behaviour the third, as stated earlier. He also observed that language began as overt expression and by being gradually internalised became silent speech and the foundation of thought.

The question naturally arises, if in fact defectives are deficient primarily in their use of words, can this deficiency be supplemented by training? To test the possibilities in a limited way an experiment was carried out which can be briefly reported. It consisted of making use of a simple conditioning technique for teaching the recognition of letters and words. Conditioning in this sense means the repeated linking together of written words and pictures. This method of teaching reading used to be quite common in schools and its use here represents not so much a discovery as a recognition of the need for continued reinforcement and practice with the severely retarded. An apparatus was constructed in which a picture could be presented which the child would name. He was then rewarded with a sweet. The next stage was to present a new picture in the apparatus—namely, a smaller version of the previous picture and accompanied by a word describing it. For example, in the first stage a picture of a cow would be presented and in the next stage a smaller version of the picture accompanied by the word "Cow". If the child said "Cow" to this picture he was then presented with the third display, which was simply the word "Cow" alone. A repetition of this series usually resulted in the pronounciation of the word when it was presented without a picture. Several words were taught in this way. The difference between each word and similar words—for example, the difference between "Cow" and "Bow"—was next learnt in a similar way, and finally a test was given. In each case it was found: first, that the severely subnormal could learn to recognise printed words in this way; secondly, that they could transfer the technique rather quickly; and thirdly, that their test results

several weeks later were better than their test results shortly after learning. This experiment seemed to offer hope of improving the verbal proficiency of the severely retarded in a lasting way and also presumably their capacity to think about problems.

The work which has been done from the Medical Research Council and the Manor Hospital in the past, of training the subnormal and the severely subnormal in motor tasks, may possibly be extended if techniques can be developed for aiding them to learn processes which they normally find difficult. In these few examples an attempt has been made to present experiments which demonstrate the capacity of defectives to perform mental operations which they are supposed to be incapable of performing. It is appreciated that this work is only a beginning, but in one mental deficiency hospital near London considerable progress has already been made in teaching reading to a group of patients of I.Q. 40-50—in other words, on the borderline of ineducability. This experience, and the experiments reported here, suggest ways in which such patients might be helped to acquire new and useful skills. Their improvement in motor skills went beyond expectation at the time. It is thought that some improvements can also be effected in scholastic skills.

SUMMARY

The traditional view is that the low-grade mental defective is relatively ineducable. It is suggested that part of this ineducability may be traced to conditions of deprivation which are not conducive to the learning of fundamental processes upon which other learning may take place. Experimental work with defectives has shown that while initial performance on tasks is related to intelligence-test performance, the final performance after training is not. The conclusion drawn from other experiments is that defectives are lacking in the controlling function of language, but even here there are grounds for supposing that a measure of verbal efficiency can be achieved.

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TEACHING IMBECILES INDUSTRIAL SKILLS

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This article briefly reviews a number of studies concerning the trainability of imbeciles, and attempts to indicate some of the practical and theoretical implications arising from this work. It is hoped that this account of some of the problems connected with the training of severely handicapped people may interest those working with the cerebral palsied.

Traditionally, the problem of industrial placement of normal people has been considered in terms of *selection* rather than *training*, and to this end a great deal of valuable research has been carried out with excellent results for the industries concerned. Inevitably this process involves the rejection of a varying number of people who fail to indicate specific aptitudes for particular jobs, the number of rejects depending to a great extent on the prevailing economic conditions. Regrettably, consideration of the problem has all too often stopped at this stage, and, among others, psychologists have been apt to make the error of supposing that mechanical and other industrial abilities are necessarily fixed and immutable. It has already been shown in many studies, for example, that this is by no means so true of intellectual capacity as was formerly thought, and therefore there is no reason to supose that other human abilities should not, within limits, change. Thus, in industrial selection the emphasis has been on the individual as he appears during a few hours of assessment, which with normal people will have considerable prognostic validity whether it be on industrial, military or educational tasks. In the field of severe mental or physical handicap, however, we start with the rejects, and the problem tends to be one not of selection so much as the investigation of what limited potentials remain to the patient, and, granted suitable training, how these can best be exploited to his advantage.

During the last century great strides have been made in the discovery of special methods of educating the blind and the deaf, while during World War II attention was focused on the rehabilitation of the physically disabled. A large number of post-war researches have indicated methods of training the mentally subnormal in institutions, resulting in greatly accelerated and increased rates of return to the community as self-supporting members of society. Part of this work has been conducted at the Manor Hospital, where rehabilitation and training schemes for adolescent and adult high-grade defectives of both sexes have been running successfully for about seven years.

In addition, experiments have been carried out on the learning abilities of adult imbeciles, whom the new Mental Health Bill describes as "severely subnormal", hopelessly incompetent patients on whom clinical opinion has been exceedingly gloomy. Clinicians have concentrated on the imbecile's disabilities as seen

on examination, and there has until recently been no systematic attempt to ascertain whether these disabilities might in some cases be modified or overcome. The clinician's view-point which, as will be seen, is in a sense both correct and incorrect, can be summarised as follows (Hermelin 1956):

(1) Imbeciles at best find it extremely difficult to concentrate, and more typically seem capable only of involuntary and momentary attention.
(2) They are incapable of comparing and discriminating between cause and effect.
(3) They are quite incapable of adapting themselves to anything out of the ordinary.
(4) They can perform only the simplest of routine tasks under constant supervision.
(5) All this makes them unable to contribute appreciably towards their own support.

The following is a brief outline of a few of the many studies summarised by O'Connor & Tizard (1956) and Clarke & Clarke (1958).

Laboratory Studies

1. *Question*: Can imbeciles respond to incentives? (Gordon *et al* 1954).

Task: A test of persistence involving sitting on a chair, holding one foot above another chair; this is an exceedingly unpleasant test.

Subjects: 27 male imbeciles, mean I.Q. (Stanford-Binet) 35.

Results: Imbeciles were able to make intense and sustained efforts, and the norms for this group were above that for normal people. They were divided into three equated groups of 9, and tested every day for ten days. The Control Group was given no incentive, the Encouragement Group was told every five seconds that they were doing well, and the Goal Group were shown a visual target, which continuously recorded their progress, and which on each occasion was set 10% higher than their last score. Final scores emerged in this order, with Controls only gaining 2%, members of the Encouragement Group gaining 46%, and those in the Goal Group gaining 115%.

2. *Question*: To what incentives do imbeciles respond best on a simple repetitive task? (Gordon *et al* 1955).

Task: To place small nails one by one into the holes of perforated zinc sheeting, mounted on a frame, using one hand only. This involves hand-eye co-ordination and some dexterity, which would in theory seem to be difficult for imbeciles.

Subjects: 40 male imbeciles, divided into 4 groups of 10, equated on the basis of initial performance.

Results: Subjects were told that this was real work and no talking was allowed. They were given a one-hour trial per day for eight days, with the following results.

Control Group: average 995 nails per hour.

Co-operation Group (2 teams of 5, co-operating within each team against the other): 1046 nails per hour.

Competition Group (individual); each subject was paired with another for purposes of competition: 1063 nails per hour.

Goal Group: 1111 nails per hour.

All these average scores were significantly different from each other, and it was concluded that imbeciles responded to incentives in an orderly and predictable way like normals.

3. *Question*: Can imbeciles learn a task involving complex spatial relationships? (Tizard & Loos 1954).

Task: Minnesota Spatial Relations Test, comprising four large form-boards. Two boards are designed for one set of 58 geometrical shapes, and the other two for a second set of 58. These shapes have to be fitted into corresponding holes in the form-boards.

Subjects: Eight male imbeciles, mean I.Q. 34.

Results: Only four could do the task on first trial, exceeding, of course, the normal time; two were trained specially and an-

other two failed altogether and were dropped out of the experiment. The six then had eight trials on each of the four boards at the rate of two trials per day. One month later all were tested again on all four boards. Results indicated that these imbeciles were able even after a month to do this relatively complex task as well as and in some cases better than the normal untrained adult. All had shown a rapid improvement with very little practice, and considerable transfer of training effects from one type of board to another.

Workshop Studies

1. *Question*: Can imbeciles do a simple industrial task and work steadily through the day? (Loos & Tizard, 1955; Clarke, 1958).

Task: The folding of cardboard food containers for a London firm. The job involves synchronous bimanual movements, speed and dexterity.

Subjects: Six male imbeciles, mean I.Q. 33.

Results: The experimenters analysed the job and the movements involved, and taught each of the six imbeciles to do the task correctly right from the start. Within two weeks they were able to make deft and precise movements, and it was established that they could do this job as well and as rapidly as feebleminded patients (average I.Q. 70) who were in training for community placement. The outcome for this group is that six years later they have become almost entirely self-regulating, needing a minimum of supervision and able to produce up to 70,000 folded boxes per week. Whereas at the beginning incentives had to be very concrete, now they can be quite remote, and the members of the group appear to be exceptionally happy.

2. *Questions*:

(*a*) Can imbeciles acquire comparatively difficult skills?

(*b*) To what extent does initial ability correlate with final level after training (as it does with normals)?

(*c*) What are the limits of imbecile trainability? (Clarke and Hermelin 1955).

Subjects: The same 6 men as in the previous experiment; all were certified as imbeciles; two could hardly talk at all; three could not name colours correctly; two were rubbish hoarders; two were unstable, one of these showing psychotic traits; four did not know their ages; two could not count at all, and only one could count above ten. Their impairment was therefore severe, and in fact they had originally been selected as being considered virtually unemployable. I.Q. range: 24-41, average 33.

Task A: Cutting insulated wire into 10-inch lengths with an error-tolerance of only ¼inch. This involves the use of a hand guillotine, and fairly dexterous movements.

Results: Two one-hour sessions were given, one week apart. Initial and final scores for a five-minute period were as follows for the six subjects:—35-46; 23-33; 40-52; 35-56; 16-48; 15-57. At the end of the second session there was no certainty that the peak had been reached but in general their level had already become similar to that of high-grade defectives, with I.Q.s 30 or 40 points higher. Most of the imbeciles could earn between £2 and £3 per week on this task.

Task B: Soldering four different coloured wires to the correct terminals of an 8-pin television plug (*see Plate*). This involves distinction of colours, appreciation of spatial relationships and the handling of solder and soldering iron.

Results: Initial scores with assistance: range 4-19 minutes per plug, average 8 minutes. Final score after 34 trials, without assistance: 1 minute 42 seconds to 3 minutes 30 seconds, average 2½ minutes. Earnings estimated as about £3 per week,

but this task is very near their limits of trainability and is not really suitable; simpler soldering is, however, appropriate. There was no correlation between initial and final status.

Task C: Bicycle pump assembly: this involves nine operations which must be performed in exactly the right order. In theory this task is difficult because a sequence has to be remembered, and unfamiliar tools such as screwdrivers used.

Results: Initial scores, with assistance: 4½-11 minutes per pump. Final scores, without assistance, after 30 trials: 54 seconds to 1 minute 50 seconds.

Mongol aged 24 yr. I.Q. 34. Soldering four different coloured wires to an 8-pin television plug.

CONCLUSIONS

From these workshop studies, three principles emerge: (1) the initial ability of imbeciles tends to be exceedingly low; (2) their initial ability has little relationship with the level achieved after training; and (3) the main distinction between the performance of imbeciles and normals on *simple* tasks is not so much the end level as the time taken to achieve it.

From this and other work several learning principles have been summarized (Clarke 1958, 1959).

(a) *Incentives.* The imbecile, like the normal person, is very much affected in the learning situation by the presence or absence of suitable incentives.

(b) *Break-down of work.* The task needs to be broken down into its basic constituents and taught in the right sequence.

(c) *Correct movements* must be insisted upon from the start.

(d) *Learning should be spaced*: many short periods are better than few long ones.

(e) *Need for over-learning.* The process should be taken well beyond the stage at which correct responses are obtained so that the response becomes deeply ingrained.

(f) *Accuracy* rather than speed should be emphasised at first.

(g) *Material* should be set out in such a way that muddle or fumbling is minimised.

General Conclusions

It can be seen from the evidence presented why the traditional clinical viewpoint has been in a sense both correct and incorrect. The picture presented by the imbecile after special training is markedly different from that when his initial difficulties are taken at face value as prognostic of *permanent* difficulties. Two general conclusions, which may well have implications for fields wider than imbecility alone, appear to be justified.

1. One may be very wrong in assuming that human qualities such as motor co-

ordination, concentration, visuo-spatial ability, dexterity, responsiveness to training or incentives, are themselves immutable. Indeed, the time has come when we need to know much more about the processes underlying such qualities and the limits to their change imposed by constitutional factors.

2. As a corollary, because of the exceptionally poor initial ability of some handicapped persons, the social or work prognosis yielded by even detailed examination may in some cases be very inaccurate. Responsiveness to training should thus be one aspect of *prolonged* assessment. Selection within a handicapped group which depends on short tests or brief periods of trial may thus yield results of far less value than corresponding methods would with normal persons.

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THE MOVEMENT OF FILMS BETWEEN COUNTRIES

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Introduction

There are an increasing number of extremely interesting and useful films in the world relating to cerebral palsy and its many facets. These films are not so widely known as they ought to be; nor are they so frequently borrowed as they might be, outside their native countries. Among the many reasons for this are the formidable formalities encountered in passing films through the national Customs barriers. The Customs of the United Kingdom are either more or less formidable than those of other countries, depending on which side they are viewed from. I hope to show here how these obstacles can be simplified and even removed altogether.

The problems encountered when dealing with films seem to multiply because the film is not yet accepted as an everyday means of communication. Problems arise from the fact that films are also a medium of entertainment, and with scientific films it is difficult to dissociate this function from their other purposes. When films are accepted as a means of communication, like books or printed papers, we shall soon see a freer flow of information. Meanwhile, most of the world's governments continue to subscribe to the ideals of Unesco Charters. Changes take place at an imperceptible rate. Nevertheless, changes do occur, and so far as the United Kingdom is concerned the last year has seen a move to make some of the procedures a little easier and less complex.

In June, 1957, the Scientific Film Association[1] published a catalogue of *Films on Cerebral Palsy*, which has now been revised as a supplement of the *Cerebral Palsy Bulletin*. The catalogue contains details of 44 films on this subject which are available in Britain, though not all are of British origin. Since 1957 many copies of this little book have been sent overseas, and many enquiries for the films have resulted. Many more people might have borrowed them if the conditions had been simpler.

Exporting Films from the U.K.

When a prospective borrower overseas wishes to borrow a film from Britain his first course, as was pointed out in the foreword to the S.F.A. catalogue, should be to try the British Council. This body was expressly set up to pass on to countries overseas information about life and development in Britain. Sometimes even the local British Embassy can be cajoled into co-operation.

If the British Council cannot help, the next step is a direct approach to the owner or distributor. In all matters connected with the movement of films, time is a vital factor—the more notice the borrower can give of his intentions the more likely he is to succeed. For the private owner in Britain faced with a request from overseas, it seems an easy matter simply to pack and post the film. This can be done and will not encounter any difficulties until

the film is *returned* from abroad. The owner will then be faced with the frustrating problem of satisfying the Customs that the film is of British origin, or, if of foreign origin, that the proper duty has previously been paid on it and not later refunded; and also that no processing has been carried out on it, or additions made to it while abroad.

It is at this point that I have received so many requests for help. The essence of the problem is one of identification: this can be made much easier if the owner follows the procedure outlined in the next paragraph *before* sending the film abroad.

For every apparent difficulty a means has been provided to make the regulations work. From all offices of H.M.S.O.[2] or any bookseller (on order), a Customs form No. 116 (Sale) may be bought for 3d., plus purchase tax and postage. After completing the necessary particulars, leaving open any doubtful questions, the owner should call in the Export Officer from the local office of H.M. Customs and Excise. The telephone book will provide its address. The Officer will come and inspect the film, probably seal the packet, and certify the Form 116, retaining the duplicate half. The owner can now either post the film, or send it by any other means. When the parcel is posted the postmaster will sign the form to say that it has left the country, the owner then sends his half to the Customs and receives the stamped copy in return. When the film comes back, ordinarily by post, the owner will receive a Notice of Arrival (C.160) from the Customs authorities at the post office. The owner should return this notice, together with the stamped Form No. 116 (Sale), which should be endorsed across its face as follows:—

> "We hereby declare that no negative or positive film has been added to the above-mentioned film while it was abroad".

The film will then be released without further formalities.

More recently a Form C.160J has been introduced by the Customs to be sent to the owner when a package appears to contain cinema films; this should provide for a simple and speedy routine.

If Form 116 (Sale) was not used when the film was exported, admission free of duty should not be claimed on Form C.160 but on Form C.179, which will be supplied by the post office in all cases to be used if necessary. Re-importation will always be made easier if the overseas sender indicates on the Customs declaration affixed to the package that the film is being returned after viewing abroad.

Films sent to this country by post must, of course, be packed in accordance with postal regulations and must be sent by an authorised post—i.e., by parcel post or letter post (or small packet) bearing an international (Customs) green label. If the film is exported and re-imported otherwise than by post, the local Customs Officer will explain the procedure.

Paradoxically, borrowing from film libraries is somewhat more difficult. Here the problems are different. Since many libraries are a commercial venture, the films they distribute represent part of their livelihood. When one of their films is sent abroad for a considerable time, they lose heavily on the loan, and their loss cannot be made good by increased hire charges because this would make them prohibitive to the borrower. Many of the larger free loan libraries, such as those associated with pharmaceutical companies, will entertain enquiries provided they are forwarded by their local agents. In either case the export procedure would be handled by the library's or company's export department, who are well qualified to deal with its snags.

Importing Films into the U.K.

As in exporting films, one of the importer's most valuable assets is time. It is useless having films reaching this country by air from the far corners of the earth in a few days if they are to be held up on arrival by a lack of the documents to facilitate their entry.

Let us suppose it is decided to view in Britain a film from abroad which will advance the cause of research here. The first step is to get the following details from the owner before shipment: the correct title of the film, its length in feet, and an invoice stating its description and value. It may also be helpful later to find out from the owner whether a certificate of the educational character of the film has been issued by the government of the producing country.

Film imported into this country is liable for duty at 1d. per foot. Under section 6 of the Import Duties Act, 1958, and paragraph 3 of its fourth schedule, application may be made to the Board of Trade[3] for a duty-free Treasury direction or "licence" in respect of goods imported for non-commercial use in the advancement of science, learning, art or sport. Details of this are contained in Notice DFN 3, supplied by the Board of Trade. From the same Department the necessary application form (D.F.A.3) can also be obtained.

Provision is also made under the Import Duty Reliefs (No. 3) Order, 1958, for relief from import duty on (*a*) films, film strips, micro-films or sound recordings of an educational, scientific or cultural character produced by the United Nations or one of its specialized agencies, and certified as such by them (full details will be found in Notice No. 370, obtainable from the Commissioners of Customs and Excise[4]); (*b*) educational films certified as such by the British Film Institute[5] on behalf of the Minister of Education. This relief depends on previous certification of the film by the government of the producing country and is also conditional on the existence of reciprocal facilities for British films imported into that country. Further details may be obtained from the British Film Institute or from the Commissioners of Customs and Excise, whose Notice No. 59 deals with this subject.

The possession of this Treasury licence does not, however, avoid the necessity for an import licence. It is still necessary to apply for an "Application for Import Licence—Exposed Cinematograph Films" from the Board of Trade[6] for all films coming from hard-currency areas, the U.S.S.R. and associated states. The need for such an import licence can always be discovered during the preliminary enquiry from the Board of Trade.

When the Notice of Arrival of an imported film is received, two further steps must be taken before the consignment can be released. For Customs purposes, duplicate copies of Form No. 22 (Sale) must be completed to accompany the entry. These forms can also be obtained from H.M.S.O.[2], price 3d. plus purchase tax and postage. In addition to answering the questions involved, this endorsement should be written across the face of the document:—

> "Claim exempt from duty under the Import Duties Act, 1932, (and/or from silk duty) by section 8, Finance Act, 1936, Licence No..... dated....."

The Form 22 (Sale), together with Form 160 and any appropriate import licence, Treasury licence, certificate, etc., should then be returned to the post office.

Except in the case of films which have to be submitted to the British Film Institute, and provided the documents have been properly completed, the package will be released by the Customs and delivered promptly by the Post Office. However, if some hitch does occur, it is always worth remembering that H.M. Customs Officer prefers to be consulted before misunderstandings arise, rather than afterwards.

Permanent Imports

In this paper I have been concerned primarily with the temporary loan of films. The procedures are much the same for the permanent import of films. If it is difficult to find the "hard currency" to pay for the film, it is possible to use Unesco *billets* as currency. Details of this scheme, with regard to films, can be obtained from the British Film Institute.'

The Transport Agent

For all the steps and procedures outlined above, except applications for import licences, Treasury directions, etc., which the importer should undertake personally, the prospective film borrower can employ a transport agent[8] experienced in the film business, who will charge about 30/-, excluding carriage, for each operation. The agent will act on his client's behalf in all matters relating to the import and export of the film, leaving the client completely free. However, this is only a question of expediency. The agent has to go through exactly the same procedures as the borrower himself, who, armed with this brief exposé, should in most respects be equally well placed.

ADDRESSES

1. Further copies of the supplement, "Films on Cerebral Palsy," can be obtained from: Scientific Film Association, 3 Belgrave Square, London, S.W.1.; *or* National Spastics Society, 28 Fitzroy Square, London, W.1.
2. H.M. Stationery Office, P.O. Box 569, London, S.E.1.
3. Board of Trade, Industries and Manufactures Dept., Div. 1, Lacon House, Theobalds Road, London, W.C.1.
4. Commissioners of Customs and Excise, King's Beam House, Mark Lane, London, E.C.3.
5. British Film Institute, Film Appreciation Dept., 4 Great Russell Street, London, W.C.1.
6. Board of Trade, Films Branch, Horse Guards Avenue, London, S.W.1.
7. The Secretary, British Film Institute, 164 Shaftesbury Avenue, London, W.C.2.
8. e.g.: Northern Transport Agency (London), Ltd., N.T.A. House, 47-48 Duke Street, St. James's, London, S.W.1.

CORTICAL LESIONS IN CEREBRAL PALSY

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MY EXPERIENCE of cerebral palsy has been largely confined to children combining this condition with considerable mental retardation and, often, epilepsy. It would not surprise me to learn that in paretic patients with more normal intelligence, lesions are more frequent and severe in subcortical formations. Cases in my collection presented at autopsy very conspicuous cortical abnormalities.

Perhaps the most common abnormality is scarring or ulegyria, which may affect virtually the whole brain (fig. 1). The gyri are atrophic, thin and hard, while the sulci are wide. This gives the brain a walnut-like appearance. The condition is particularly obvious in histological sections (fig. 2) stained by the Holzer method for fibrous glia. One sees, on higher magnification, cellular glial hyperplasia with a dense network of glial fibres. The cells are mostly astrocytes (fig. 3), but may be atypical (large or multinucleated) or lie in syncytial groups. In general the greater the hyperplasia the more atypical the cells. Fibrous gliosis may be very diffuse, affecting the whole thickness of the cortex. However, it often tends to form a pattern. Marginal gliosis, as in fig. 4, is one of these patterns. The fibres are particularly

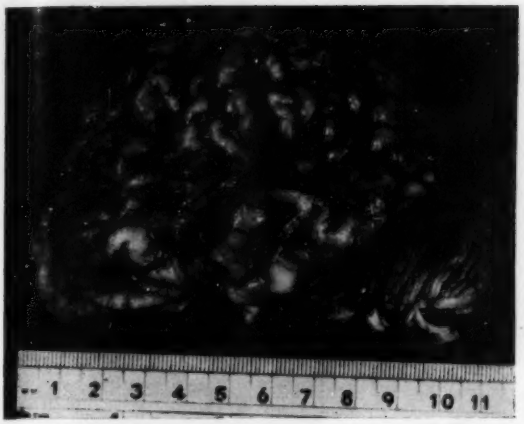


Fig. 1

Fig. 2

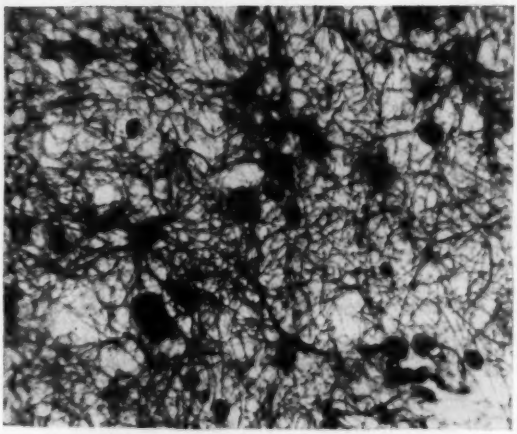


Fig. 3

Fig. 4

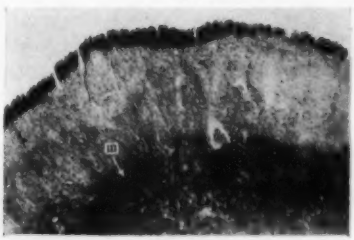


Fig. 1. A micropcephalic brain weighing 315 g. and showing ulegyria.

Fig. 2. A coronal section of the frontal lobe of a ulegyric brain. Holzer stain x 1¼.

Fig. 3. Fibrous gliosis with astrocytic hyperplasia. Holzer x 550.

Fig. 4. Fibrous gliosis affecting the marginal and third layer of the cerebral cortex. Holzer x 20.

dense in the subpial layer of the cortex. This is often combined with gliosis in, roughly speaking, the middle of the cortex i.e., in the third layer (fig. 4). There is a varying degree of neuronal loss. In extreme cases scarcely any nerve cells are left. In general, superficial layers seem to be more vulnerable than deeper ones. This loss of nerve cells may be widespread and fairly uniform, as in fig. 5, taken from a case of sequelae of kernicterus due to Rh factor incompatibility. I should mention that there was only a slight degree of fibrous gliosis in this case, and that the basal ganglia are also affected in these cases. Cellular loss may, on the other hand, be focal, or rather multifocal, as in the example fig. 6. This is the cerebral cortex of a child who developed normally up to the age of 6 months. He then had severe intractable gastro-enteritis lasting 3 weeks. The child died 2 years later, and

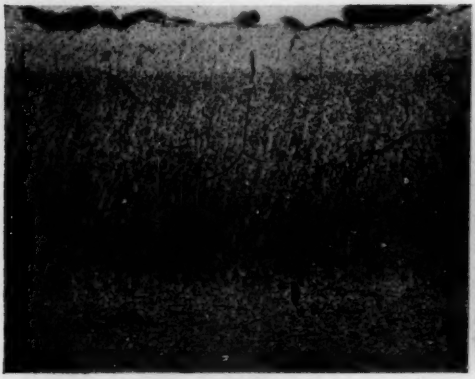


Fig. 5. Diffuse neuronal loss in the superficial layers in a case of residual kernicterus. Cresyl violet x 20.

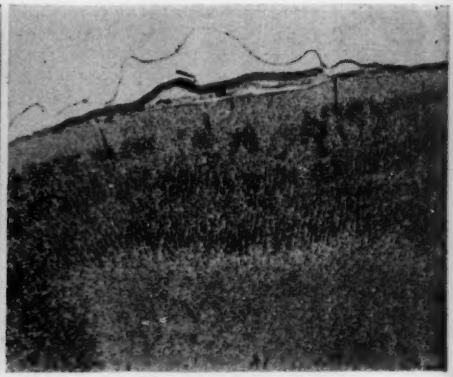


Fig. 6. Multifocal neuronal loss in the superficial layers of the cortex. Cresyl violet x 15.

the cortex showed this "moth-eaten" appearance. A possible pathogenetic mechanism in this case is marked hæmoconcentration with vascular stasis, leading to focal anoxia.

There is another form of ulegyria in which the gyri are not particularly shrunken. They may in fact look normal but are very hard to the touch. One of the cases with this condition had cerebral palsy and intractable epilepsy. Part of the frontal lobe was resected at 7 months and examination of the material resected at the time of the operation showed no significant abnormality. The epilepsy remained unrelieved and the child died 2 years later. The cortex showed marked loss of nerve cells in the superficial layers. A special feature of this type of sclerosis is the presence of large abnormally orientated nerve cells lying among hyperplastic gial cells (fig. 7). When impregnated with silver, these cells show stout processes. In this condition there is dense fibrous gliosis often particularly marked in layer 3.

Ulegyria is often associated with cavitation. This is hardly surprising since gliosis is probably in the majority of cases the result of a repair process. Repair is often incomplete and cysts are left. In fig. 8 the association of cysts and ulegyria is seen in the brain of one of a pair of impacted twins (the other died at birth). This patient was a hemiplegic epileptic who lived for 3 years. The coronal block of the brain shows also granular atrophy —a term applied to the puckering and unevenness on the surface of the gyri. (fig. 8).

A histological section of the brain of a patient with lobar atrophy, ulegyria and cavitation is shown in fig. 9. One of the occipital lobes was very hard, and on the cut surface there was blurring of the normal demarcation between white and grey matter. Small cysts were also present. This lesion was probably produced by birth injury; the case history was consistent with it. Fig. 9 shows the junction of the glial scar tissue and more intact cortex. Some of the cysts contained intracellular and extra-cellular fat (fig. 10).

Not all cases of cavitation are congenital in origin. Fig. 11 is an example of softening and cavitation due to arteritis in the case of a tuberculous meningitis treated by streptomycin. The arteritis involved the anterior cerebral artery, and resulted in a hemiplegia. Fig. 12 is an-

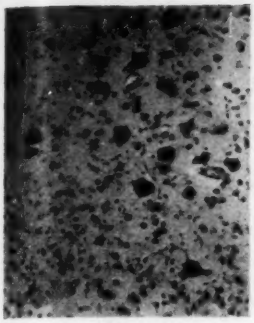


Fig. 7

Fig. 7. Abnormally orientated giant cells in a case of gliosis with giant cells. Cresyl violet x 260.

Fig. 8. Coronal block of the brain of one of impacted twins. The upper part shows ulegyria and cavitation.

Fig. 9. Junction of scar tissue and more intact cortex. Thionin x 90.

Fig. 10. Cavities in cortex containing intracellular and extracellular fat. Scarlet red and hæmatoxylin x 140.

Fig. 11. Softening of left frontal lobe in a case of tuberculosus meningitis treated with streptomycin.

Fig. 12. Embolic infarction of area supplied by left middle cerebral artery in a case of mongolism with Fallot's tetralogy.

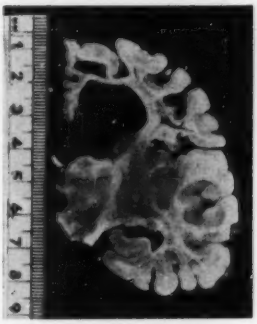


Fig. 8

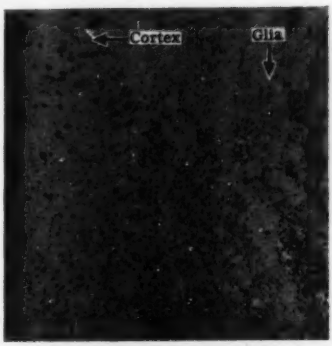


Fig. 9

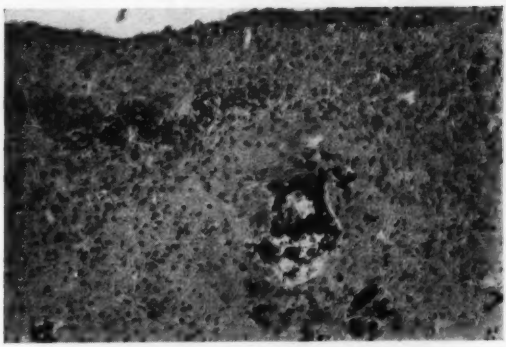


Fig. 10

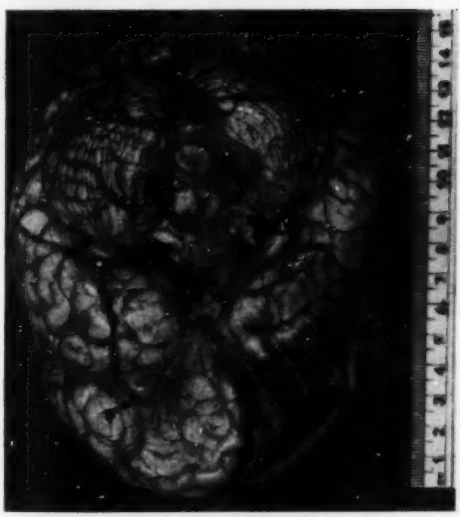


Fig. 11

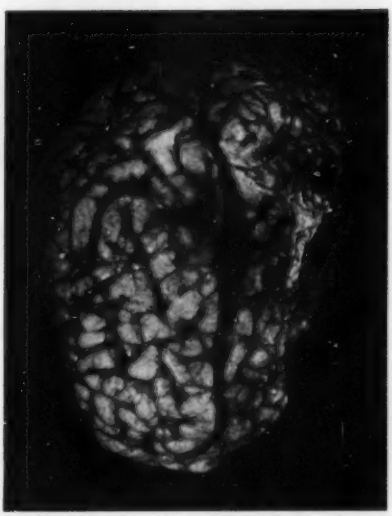


Fig. 12

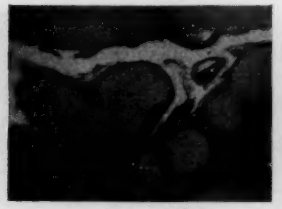


Fig. 13

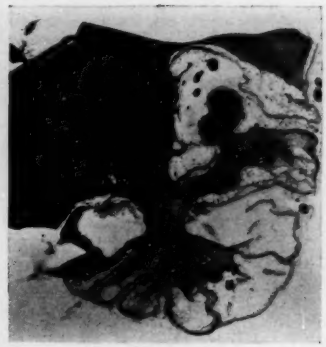


Fig. 14

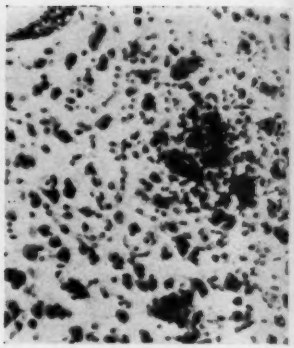


Fig. 15

Fig. 16

Fig. 17

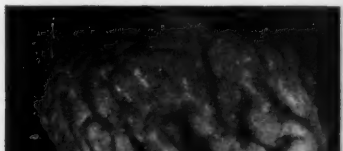
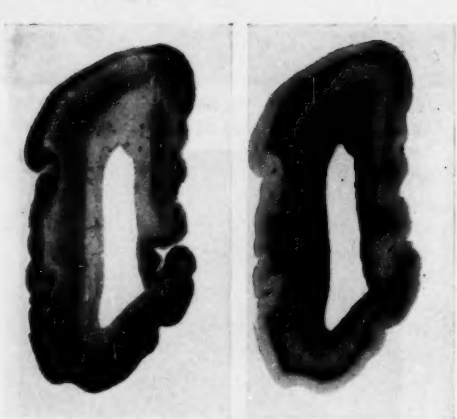


Fig. 19



A Fig. 20 B

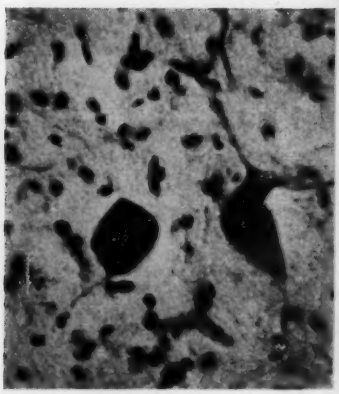


Fig. 21

Fig. 13. Plaques fibromyéliniques of the cortex. Phosphotungstic acid and hæmatoxylin x 20.

Fig. 14. Cavitation of right temporal lobe. Phosphotungstic acid and hæmatoxylin x 1¼.

Fig. 15. Perivascular cuffing and focal microglial proliferation. Cresyl violet x 140.

Fig. 16. Porencephaly with microgyria round the edges of the cavity. Cresyl violet S/S.

Fig. 17. A convoluted form of microgyria. Cresyl violet x 3.

Fig. 19. Almost total agyria.

Fig. 20. Pallial lamination of the brain in pachygyria. (a) Cresyl violet, (b) Heidenhain stain for myelin. Note characteristic 4-layered lamination.

Fig. 21. Distended nerve cells in a case of gargoylism. Cresyl violet x 350.

other example of extensive softening due to embolism of the middle cerebral artery. Such embolism with ensuing hemiplegia is not uncommon in patients with septal cardiac defects. It can occur in patients with mongolism, who frequently have such congenital heart defects.

A few words may not be amiss about hypermyelination or *plaques fibromyéliniques*. This is usually associated with fibrous gliosis of the cortex as in granular atrophy (fig. 13). This condition frequently affects the basal ganglia.

It is fair to say that the ætiology of cavitation and of sclerosis is quite obscure in most cases. The next example, fig. 14, is from a case showing bilateral cavitation and sclerosis of the temporal lobes. The patient was a boy with normal family and natal history. He fell on his head when he was 2 years old, remained unconscious for one hour, and had two fits the following day. His development after that was somewhat retarded. At 4 years he had 3 further fits and meningitis with raised protein in the C.S.F., which also contained 80 mononuclear cells per cu. mm. In spite of a thorough investigation in one of the leading London hospitals, no cause for the condition was found. After the illness he developed severe epilepsy and had cerebral diplegia. He died at 11 years. You can see one side of the brain with the lesion in fig. 14. This was not strictly confined to the temporal lobe, but extended also to the neighbouring parts of the brain. A Nissl picture in one of the less affected parts showed cellular loss in the 3rd layer. In the more severely affected areas there was confluent cavitation in the 3rd layer. Elsewhere there was perivascular cuffing and focal collections of mononuclear cells (fig. 15). The cause of this condition is unknown. Was it an infection? Did it start with the trauma as suggested in the case history? Was epilepsy a contributory factor? These are the questions to which we have so often no definite answer.

Most of the cases in my material are, however, clearly congenital in origin, and often pre-natal. This refers, for example, to porencephaly. In fig. 16 porencephaly is associated with microgyria. And this

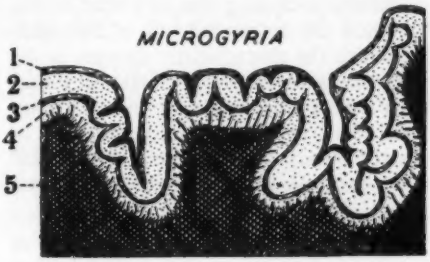


Fig. 18. Schematic representation of the cortex in microgyria. Layer 3 contains myelin with few nerve cells.

brings me to the consideration of microgyria and pachygyria. An example of microgyria is also seen in fig. 17. It shows a very convoluted arrangement of the cortex. The arrangement can be disentangled in some of the less convoluted portions of the cortex, to show the characteristic lamination in 4 layers (fig. 18). Contrariwise, cases of pachygyria show lack of gyri (fig. 19). And agyria is an extreme degree of this condition. Histologically, however, there is some similarity between these cases. In fig. 20a, an example of pachygyria, a four-layered cortex can be seen, and the myelin picture of the same brain (fig. 20b) confirms this similarity even more strikingly. A narrow layer of myelin occupies the third layer of the pallium.

It has been suggested by Bielschowsky that these conditions are caused by some impediment of cellular migration from the periventricular matrix to the periphery during the early months of intrauterine life. As a result some of the cells fail to reach the cortex, remaining as a wide heterotopic formation—layer 4 of the microgyric and pachygyric cortex. They are separated from the cortex proper by the myelin-containing layer 3, which is analogous to the U-fibres of the white matter. It is further suggested that pachygyria, and even more so, agyria, are due

to such an impediment in the migration at an earlier stage than in cases of microgyria.

Before concluding this very brief survey I should like to remind you of the lipidoses with the characteristic changes in the nerve cells of the cortex and elsewhere. It is perhaps not quite so widely known that some nerve cells in gargoylism or lipochondrodystrophy present similar cellular changes, as in the example shown in fig. 21.

There are many more examples of cortical lesions in cases of cerebral palsy. These will suffice, however, to demonstrate the great variety of such lesions. I ought to add that I did not make a special study of the relative incidence of the lesions in different parts of the hemispheres. I do not know whether lesions in the frontal lobes are more common than in the occipital or parietal, and so on. I should think that such a study would be very difficult and inconclusive, because hardly any part of the cortex has been normal in most of my cases, though the severity and character of the lesions have varied greatly in the same brain.

Some of the lessons to be learned from the morphological study of these cases are as follows:—

1. In a few cases it is possible to determine the ætiology of the condition, e.g., birth injury, meningitis, embolism and so on.

2. In some other cases it is possible to set the time limit for the onset of the causative disturbance, e.g., microgyria, pachygyria, or less certainly ulegyria.

3. In most cases even this is impossible without access to carefully and thoroughly recorded clinical notes. I have been very fortunate in this respect in having at my disposal clinical information compiled by clinicians. I would like to take this opportunity to express my indebtedness to them. Provided that we can have such co-operation in the future, I believe that neuropathology can contribute significantly to the study of cerebral palsy.

ACKNOWLEDGEMENTS

I am indebted to Messrs. J. & A. Churchill for permission to reproduce Figs. 3 and 13, which I had originally published in Hilliard and Kirman's "Mental Deficiency". The editors of the following periodicals have also kindly allowed me to reproduce illustrations previously published in their journals: *Journal of Clinical Pathology*, fig. 2; *Journal of Pathology and Bacteriology*, figs. 16, 17, 18, 19 and 20; and *Journal of Mental Science*, figs. 4 and 13.

RESULTS OF CEREBRAL SURGERY FOR MOTOR DISORDERS*

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This short contribution deals with the recent work on the relief of tremor and rigidity in adults by cerebral surgery, and some of the facts and speculations that surround it. No operation as yet relieves the spasticity of cerebral palsy in childhood.

For the past four or five years some neurosurgeons have been making destructive lesions deep in the cerebral hemisphere and claiming that this procedure relieves the symptoms of tremor and rigidity without adding any motor deficits —in particular without imposing even a mild hemiplegia. Irving Cooper, working at St. Barnabas Hospital, New York, began to produce destruction of tissue by occluding the anterior choroidal artery which supplies a variable amount of the basal ganglia, internal capsule and thalamus. Finding the effects of this too variable, he then changed his technique to produce destruction by the injection of alcohol; other surgeons use electro-coagulation or even a leucotome. All are guided by landmarks taken from X-ray shadows of the ventricles, but now that more is known of the amount of individual variation in anatomy no-one is making confident claims to accurate localisation in a particular nucleus or tract in a given case.

Probably between 1000 and 2000 cases have been operated on. Cooper himself has now done over 500 cases. I do not intend to review his work, or to assess the contribution of others such as Bucy, Walker and Meyers in America, or Fenelon, Guigot, Hassler, Reichert and Lexsell in Europe, or to discuss the general problem of the therapy of tremor and rigidity in the adult. My own experience, which has been gained through the help of Mr. McCaul at the Whittington Hospital, is trivial in comparison with Cooper's, but is quite sufficient to convince us that in some cases long-standing tremor and rigidity can be relieved immediately, and so far permanently, without disturbing the patient's ability to perform fine finger movements. This is the essence of Cooper's claims, and many other centres have confirmed it, so it is now beyond dispute. There is dispute, however, about the proportion of clear successes and the incidence of complications. Several centres have naturally tried it for various spastic and athetoid conditions in childhood, but so far without any success. This failure to affect spasticity as distinct from rigidity may give a clue to the objective nature of the physiological difference between them. Successes have been claimed in some cases of dystonia musculorum deformans in childhood, and hemiballismus.

Implications

Before we consider the implications of this discovery the following additional facts about the procedure may deserve consideration.

*Read at the N.S.S. International Study Group on Child Neurology and Cerebral Palsy, Oxford, September, 1958.

(1) The operation has little or no beneficial effect on speech, immobility of the facies, akinesia of the limbs, or any of the autonomic manifestations of Parkinsonisim. It affects only tremor and rigidity of the contralateral side.

(2) The effect of the lesion on tremor and rigidity can be immediate—i.e., within 5-10 sec. in favourable cases. A temporary disturbance may be brought about by injection of local anaesthetic or distension of a small balloon at the end of a cannula, and this is the base of a clinical test to guide the location of the lesion.

(3) Electrical stimulation at the site before making the lesion may augment or abolish tremor and sometimes has the effect of lessening the rigidity. The effects of stimulation and also recording from the region in which lesions are made are variable and no-one has yet linked the results to success or failure at the operation. But this is an interesting field for further investigation.

(4) No-one yet has post-mortem material from representative successful and unsuccessful cases, and so no-one knows for certain what structures are involved or even whether the lesions are large or small. This uncertainty at the moment clouds all discussion. For descriptive convenience one lesion of choice is called "pallidal" and another "thalamic," but we should guard against letting these terms influence our thinking.

Perhaps the first effect of this discovery is to emphasise a need for more honesty than heretofore about our basic ignorance of the mechanism through which the efferent side of the nervous system operates, for this work is based on pure clinical opportunism. No-one can claim that it followed by logical deduction from any prior observations or even any speculative scheme of organisation of the motor system. In view of the various crude and blind procedures that are being used, the immediate reaction of most people is surprise that complications are not more common. For example, the thalamic syndrome has never been seen by Cooper in about 300 cases of inflicted thalamic damage, which he does not claim to be consistently accurate in location. It is most improbable that the lesions are sparing fibres of the internal capsule, but hemiplegia occurs in all series to an extent of only 1 or 2 per cent. There is a tendency now to assume that spastic hemiplegia occurs only when a large blood-vessel is interfered with and to acknowledge that we are ignorant of the essential lesion in nervous tissue that gives rise to this familiar picture.

Since in these and other ways our expectations have not been fulfilled, people have naturally been wondering how to explain the result and how to modify and extend our notions.

Six Suggestions

The first fact to which attention is called is that a surgical lesion is relieving a symptom when it is placed in the presumed area of causal pathology. Meyers is now aiming to place his lesion in the substantia nigra and is claiming consistently good results. One might reasonably have expected all the symptoms to have been exacerbated in a proportion of cases. In this context we might recall an apparent phenomenon of cerebral function already suggested by observation in another field—namely, in epileptic surgery and in particular in hemispherectomy. The normal hemisphere may show gross electrical abnormality which disappears immediately the diseased hemisphere is removed. The phenomenon is epitomised in Penfield's phrase, "Bad brain is worse than no brain." The inference is that malfunctioning nerve cells in a region of disease can cause disturbance in function in normal healthy nervous tissue at a distance. Acting on this hypothesis it becomes reasonable to expect improvement if one completes by surgery a process that Nature is taking too long over. On this view, then, the Parkinsonian syndrome does not represent a deficit so

much as a disturbance of normal function by malfunctioning but essentially dispensible nervous structures.

As a second suggestion, some hold that since excision of excitable motor cortex temporarily alleviates tremor, the effects are produced by disturbance of the cortico-spinal pathway. On this view it would be logical to aim a small isolated lesions at the genu of the internal capsule, where these fibres are found. Since the operation is successful without impairing fine finger movement the classical role of this pathway would need to be refashioned. But a greater difficulty is that it does not account for the relief of rigidity.

A third speculation centres round the unanswered question of what would be the deficit if this operation were performed on a group of normal persons. There might be none, but among other possibilities there might be a loss of tone and impairment of position sense on the opposite side. The normal might in other words show slight signs of cerebellar or occasionally frontal disease. To account for these signs one might invoke damage to fronto-ponto-cerebellar fibres in the anterior part of the capsule, or derangement of that part of the ventral nucleus of the thalamus into which fibres from the cerebellum run. This suggestion meets the problem of the absence of motor deficit following operation by implying that what we are calling relief of rigidity in these cases may in fact be merely a superadded hypotonia of cerebral origin overriding the rigidity.

A fourth speculation involves changes that might be brought about in the functioning of the brain-stem reticular system. This system has recently been noticed as a cortical "alerting" mechanism, but so far as we know, the basal ganglia have no direct connection with the bulbo-spinal motor centres save via the reticular system and the reticulo-spinal pathways, and the reticular system is well placed to play a significant part in movement co-ordination and the maintenance of muscle tone. Parkinson's syndrome is in part a disorder of muscle tone in the waking state only and a disturbance of the influence of attention on motor performance. Could not both these elements be looked on as the clinical signs of a deranged reticular system? It might be that, by producing further destruction of nervous tissue which is having a disturbing effect on the reticular system, some balance is restored.

A fifth speculation is that the operation is not specific with regard to the structures involved. In other words that a proportion of the Parkinsonian population will benefit from any sufficiently severe intracranial insult. This view is an extension of similar views held by some in regard to relief of focal epilepsy by surgery. Parkinsonism is much more unremitting than epilepsy but the degree of tremor is often very variable and there is evidence that in some advanced cases even the rigidity is essentially a reversible condition. Cases occur with striking remission of rigidity for a few minutes on waking in the morning, and in others there has been a great increase in mobility following E.C.T. But these are exceptional, and the percentage of successes from the operation is now reported to be so high that this is unlikely to be the whole truth. Furthermore, the first operation may completely fail and a second succeed when the lesion is differently placed.

Lastly, Cooper himself has stated that his original technique of clipping the anterior choroidal artery was an attempt to block out a functional unit. The idea behind this is that as the nervous system develops along with its blood-supply, tissue in the same territory of supply has a similar functional role. A difficulty in accepting this seems to be the individual variability in the territory which the vessels supply. If this idea is pursued it would follow that the same nervous

structures would have different functions in different individuals. This is more than most people would swallow, but such is our ignorance that this is in itself no reason for rejecting it.

The present view of the organisation of the motor system is substantially that proposed by Ferrier in 1886. That this is in need of drastic revision has been obvious, but new facts—i.e., facts unknown to Ferrier and his contemporaries — have been very few. This chance discovery is an important addition to our knowledge, but it will make its full contribution only when the post-mortem material is studied.

SUMMARY

Following a chance observation, it has been discovered that the destruction of nervous tissue deep in the cerebral hemisphere can relieve tremor and rigidity on the contralateral side in some cases of Parkinson's disease, without impairing motor performance.

There are many possible explanations for this, and six are briefly reviewed here, but the full significance of this knowledge will only be realised when post-mortem studies of the lesion in successful and unsuccessful cases are available.

A NOTE ON THE CLASP-KNIFE PHENOMENON*

RONALD MAC KEITH, D.M.(Oxford), F.R.C.P.

When I have thought of the clasp-knife phenomenon, I have thought of passive movement of stiffly extended spastic lower limbs in a diplegic or paraplegic cerebral palsy child. Perhaps I had in mind what Purves Stewart and Worster-Drought say of "clasp-knife" hypertonus:

> "Pyramidal hypertonia is of the 'clasp-knife' variety—i.e., when the rigidly extended lower limb is passively flexed at the knee . . . resistance is encountered until flexion is almost complete, when it suddenly 'gives,' as in the shutting of a clasp-knife."

I would have said that the "give" may be either early in flexion, or when the flexion is almost complete. The *Concise Oxford Dictionary* defines a clasp-knife as a "folding knife with a catch which fixes the blade when open," which seems compatible with my association of the phenomenon with attempting to "close" the "fixed" or hypertonic lower limbs.

Recently, however, conversation with a distinguished neurologist revealed that in speaking of the clasp-knife phenomenon he had in mind, as a typical example, the hemiplegic's upper limb, already in flexion. He pictured himself extending the flexed elbow and observing first an increase of tone and then a giving way. He said, "it is just like a clasp-knife—when you are opening it there is an increase of resistance."

The description in Elliot, Hughes and Aldren-Turner's book (1952) seems to fit in with this idea. They say:

> "The Spasticity" . . . (the increased resistance to passive movement) "is of the clasp-knife variety—i.e., attempts to overcome it evoke an increase of tone, but the resistance suddenly gives way when greater force is used."

Like Purves Stewart, these authors speak

*Revised from the paper read and discussed at the N.S.S. International Study Group, Oxford, September, 1958.

of already existent hypertonus, but they add the idea of an initial increase of resistance.

What Walshe describes is closer to what my neurologist had in mind, for Walshe does not mention any pre-existing, initial hypertonus.

"The presence of increased tone (in lesions of the upper motor neurone) can most readily be detected by passive stretching of the muscles, when a clearly increased resistance can be felt. This has the following qualities, most easily detected in the knee extensors. If the thigh rests upon the examiner's left forearm, placed under it as the patient lies in bed, and the right hand grasps the leg just above the ankle, on flexing the leg at the knee a few degrees of movement are easily carried out without marked resistance, but once this range is passed an active resistance is felt to develop quickly in the quadriceps, and more force is needed—considerable force in some cases—to continue the flexion. After another 30 degrees of flexion the resistance melts quickly, and the rest of the movement is relatively easily carried out. This sudden waxing and then waning of tone in the hypertonic limb has been spoken of as 'clasp-knife rigidity.' In the upper limb passive extension at the elbow will reveal a similar state."

SUMMARY

I conclude that there are two variants of the clasp-knife phenomenon: (a) when already hypertonic muscles "give" suddently on passive movement; and (b) when muscles, whether originally in hypertonus or not, develop an increase of hypertonus when the limb is passively moved and this "catch" or newly developed hypertonus "gives" on further passive movement.

The give is sometimes fairly early in passive movement, when it is a reflex response to increased tension in the muscle, or it may be late in the passive movement when it is a response to lengthening of the muscle.

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HYPERTONUS AND PHYSICAL THERAPY*

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Hypertonus and Physical Therapy

This paper will be confined to the effects which physical therapy may have on hypertonus of muscle and thus on the function of a limb or other body part. In cerebral palsy, this form of treatment has a big part to play where there is loss of joint sensation, ataxia, unwanted movement, etc., but these aspects of treatment will not be dealt with here.

As in other pathological conditions, the aims of physical therapy when applied to hypertonus are to improve function to the greatest possible degree and to prevent deformity. It is obvious that this form of therapy cannot cure hypertonus but it can ameliorate its degree. Moreover, physical treatment can modify the disability which the hypertonic muscles produce in the function of the limb or body part which contains them.

In the hypertonic types of cerebral palsy, experience shows that the degree of increase of tone with its concomitant muscle weakness varies enormously from case to case. All degrees are found from a little stiffness of the calf muscles (causing a minimal difficulty in dorsiflexing the foot) to a severe condition where whole muscle groups are wasted (i.e., have never matured) with the consequence that the limb is small and its function negligible. It seems, therefore, that most of these lesions are only partial, which may account for the almost total recovery occurring in the mildest cases without specific treatment. Thus the improvement to be expected from physical therapy largely depends on the severity of the initial pathological process.

The possible effects of physical treatment can conveniently be considered under two headings: (1) its effect on hypertonus of a muscle or muscle group; or (2) its effect on the modification of function produced by the increased muscular tone.

1. Effect of Physical Therapy on Hypertonus or Muscle

For many years, *stretching* of hypertonic muscle has been the method of choice, as indeed it still is. It is in the actual stretching procedure where advances have been made and this provides the most fruitful field for discussion and divergence of opinion.

At one time the stretching was done by hand. By its very nature this could not be applied for an adequate time and it was gradually found to be inefficient and too tiring for the therapist. It is now only considered of value in the mildest cases or where stretching by other and more efficient means is impracticable.

Plaster casts provide a most efficient method, but when the hypertonic groups are in the legs (as often happens), the cast hinders movement of the limb and cannot be kept applied long enough fully to achieve its object without the risk of harm to skin; subcutaneous tissues, and other structures.

*Read in absentia at the N.S.S. International Study Study Group, Oxford, September, 1958.

Night splints are another good way of gaining the same object, but, in an appreciable number of cases, the hypertonus is considerably lessened in sleep, and thus it might be better to allow the child to rest untramelled.

Walking calipers are probably the most efficient way of stretching hypertonic muscle. In all but the worst cases, the child can learn to walk in his calipers and thus stretch his own muscles. It is, perhaps, still necessary to point out that walking calipers *must* have knee hinges which can be locked and unlocked at will. If a rigid iron is used, the gluteal and lower back muscles become stretched when the child sits in a chair. This is most undesirable since these muscles are either normal or *hypotonic*, and stretching them will only increase their weakness.

It can be said with confidence that protracted stretching of this kind continued over years does lengthen a hypertonic muscle or muscle group, thus allowing the antagonist groups to work to better advantage and to benefit from physical therapy designed to improve their power. The *degree* of hypertonus is also modified in all cases (save where permanent contracture is present in the more severely affected) but obviously the hypertonus does not disappear and an ordinary tendon reflex is still exaggerated, even in cases where the original handicap was not great and treatment has been judged successful.

Other methods used in physical therapy have their place mainly in a relatively minor role. It is well known that hypertonus is increased by cold. Thus heating a limb can be beneficial by reducing hypertonus in the affected muscle groups, so allowing the antagonists to benefit from physical treatment designed to increase their power. In severe cases, where there is reduction in muscle bulk, some say that manual kneading of the muscle will increase the blood-flow, so helping the physiological processes concerned in muscle growth. This is probably true, but the treatment must be given often and over a long period for any effect to be expected, and there is grave danger of increasing the tendency to deposit fibrous tissue, which is latent in all but the mildest cases. This method is, therefore, to be deprecated except in the most skilful and experience hands.

Another method much in use is to harness the association between posture and the various righting reflexes to cause limb movements. In some cases this undoubtedly increases limb function, but its effect on hypertonus per se is questionable.

2. Effect of Physical Therapy on Modification of Function produced by Hypertonus of Muscle

Interference with limb function caused by hypertonus of a muscle or muscle groups is classically seen in the leg. Usually the plantar flexors of foot (gastrocnemii and soleus) are hypertonic in cases of spastic cerebral palsy. This results in a more or less permanent plantar-flexed position of the foot, which may have been present since birth. This, in turn, prevents contraction of the foot extensors, which thus become progressively weaker (stretching of a hypotonic muscle causing further weakness), and in severe cases their bulk is considerably reduced. Physical therapy can remedy this situation by putting the foot in mid-position by means of calipers (vide supra) where necessary and by increasing the power of the foot extensors by appropriate exercises.

In the thigh both extensors (quadriceps) and flexors (hamstrings) are often affected, but the flexors are frequently more hypertonic, giving rise to a similar situation to the above, with a more or less permanent flexor deformity at the knee and weakened quadriceps.

In the upper limb, the flexors and rotators of the forearm are usually the more hypertonic. Splinting in this instance is, as a general rule, impractical,

and so recourse has to be made to manual stretching of the affected muscles with exercises to the unaffected or less affected groups.

In many cases the hypertonus seems to be selective. For instance, in the foot, extensor hallucis longus is often more powerful than tibialis anterior, though both have been weakened by the process mentioned above. In these cases, physical therapy can help by substituting as far as possible the function of the former for the latter. This "replacement therapy" can be applied in other situations.

Finally it must be remembered that hypertonus is accompanied by weakness in the same muscle. In other words, it is in cerebral palsy, a spastic *paresis*. No amount of physical therapy will abolish this weakness, but, when the muscle has been lengthened by the methods already described, it is possible to improve power by the appropriate exercises.

SUMMARY

Physical therapy can modify hypertonus per se.

It can to a variable extent modify the effect that the hypertonic muscle has on limb function.

A clear distinction should be drawn between the paresis of hypertonic muscle and the weakness of normal muscle, and the increased weakness of hypotonic muscle when stretched for long periods. Where there is increased tone, stretching helps. Where there is decreased tone, stretching is harmful.

Physical therapy can increase the power of hypertonic muscle once the hypertonus has been modified.

Discussion of Dr. Crosland's Paper

DR. WILLIAM DUNHAM (*London*).—The object of our efforts in diagnosis and in disentangling the physiology of these problems is to help the child with cerebral palsy and the adult with spasticity to overcome their helplessness, by putting them in a better position to function efficiently despite their condition. We badly need a change of attitude in looking at the function of the nervous system. One example is brought out by Crosland in his paper—namely, that a spastic muscle is a weak muscle. The muscle contracts strongly against you if you try to stretch it, but from the functional point of view the muscle is ineffectual. For example, put a child with thigh adductor spasm on his side and lift the lower limb which is on top out of the way. It you now ask the child to adduct the remaining lower limb off the couch, he cannot do it, as regards active voluntary movement.

This brings me to the important point I want to make. There is another method of lengthening muscles. Whether we are holding them tight initially or not, we lengthen our muscles by contracting their antagonists. Active movement is a compound of producing a movement that you want and stopping the contraction of muscles which, except in so far as they are required for steadying, are antagonistic to that particular movement. And this lengthening by inhibition, which is a secondary consequence of active movement, has often been overlooked in the past. In *passive* lengthening by stretching one actually excites contraction (through the stretch reflex) of the muscle lengthened. On the other hand, in *active* movement one inhibits the muscles that are being lengthened. If we can teach a child to produce active movement in any circumstances, surely we should go for that,

because in so doing we are also producing the inhibitory counterpart. In these circumstances we have both the movement and the lengthening that we want, while the child is learning control simultaneously, and if this is done in functional activity the child is learning skill at the same time. Stress effort (asking the child to tackle something which is beyond his relatively small ability) does not produce this kind of co-ordinated contraction-relaxation pattern.

We must consider very seriously the use of our neuro-physiological knowledge in manipulating the central nervous system.

BOOKS — New and Not So New

L'EQILIBRE MENTAL. By Dr. Cyrille Koupernik. Librairie Arthème Fayard, 18-20 rue du Saint-Gothard, Paris XIVe. Pp. 118. 850 francs.

The brilliance of the first two volumes, *"L'Equilibre Mental"*, by Dr. C. Koupernik, and *"L'Appareil Digestif"* by Dr. J. R. Gosset, in a new series, *"La Maladie et Nous"*, forming part of the publisher's "encyclopaedia for everybody", casts a shadow of dark britannic envy on your reviewer.

The preface of the series starts with: "You have called in your doctor. He has been. He has reassured you. But you have not had from him all the explanation you had awaited . . . You would have liked to know something more, and you were right."

Dr. Koupernik's volume deals with the development of the personality from infancy to adolescence, and with heredity; with the neuroses and with mental illness; and finally with certain methods of treatment. This is an inescapable and obvious curriculum, but the reader who has tried to learn the same subjects elsewhere will acquire, from the presentation they get here, a motivation to learn that will make all the difference. It is not only that Dr. Koupernik writes concisely, with common sense and in excellent style, easily readable for anyone who has passed the G.C.E. —that would be expected if one knows Dr. Koupernik or has heard him speak. But the format, the printing and the illustrations add further life and freshness to the text. Would that there were similar books published in the United Kingdom.

R. C. MAC KEITH

ABSTRACTS

IN COLLABORATION WITH "*Abstracts of World Medicine*," PUBLISHED BY THE BRITISH MEDICAL ASSOCIATION

Electromyographic Studies in Cerebral Palsy
M. TURNER, M. A. PERLSTEIN, and H. ELAM. *American Journal of Physical Medicine* Dec., 1958, **37**, 302-326, 19 figs., 34 refs.

Clinically the motor dyfunction in cerebral palsy may be classified as due to: (1) spasticity, (2) dyskinesia (including athetoid movements, rigidity, tremor, dystonia, and ballismus), and (3) ataxia. The study here reported, from Cook County Hospital and the Paediatric Department of the Northwestern University Medical School, Chicago, was designed to determine whether cerebral palsy could be classified according to electromyographic (EMG) criteria and whether each of the above types had a characteristic EMG pattern. Emphasis was placed on patterns of movement involving several synergistic muscle groups, since previous studies of single muscles had not been rewarding.

The subjects included 38 patients with cerebral palsy (1 with the double lesion of spastic paraplegia and athetoid quadriplegia) and 10 healthy controls. The EMG was recorded by means of an Offner 8-channel ink-writing machine, with specially designed surface electrodes placed over the flexor and extensor muscle groups in the proximal and distal segments of the more severely affected limbs. In some instances EMG recordings were taken with unipolar or concentric needle electrodes and a Meditron electromyograph. Patients were examined in the supine position and recordings were taken at rest, and in response to deep tendon reflexes, cutaneous plantar stimulation, support reaction, crossed nociceptive reaction, synkinetic or associated reaction, and tonic neck reflexes. In 11 patients these procedures were repeated 20 minutes after administration of a muscle relaxant, 5 patients receiving meprobamate intramuscularly and 6 dicyclopropyl ketoxime by mouth.

From the findings the authors conclude that characteristic EMG patterns at rest and in response to various stimuli occur in the different clinical types of cerebral palsy. A tentative EMG classification of cerebral palsy is given in the following table.

	Normal and Ataxic	Spastic	Athetoid	Rigidity	Tremors
Rest activity	–	±	++	++*	++†
Spread of deep tendon reflex	–	++	±	+	–
Support reaction	–	++	–	+	–
Plantar stimulation response	+	++	±	?	?

* Simultaneously in agonist and antagonist.
† Mainly in flexors.
– absent; ± present at times; + present constantly; ++ exaggerated.

The EMG responses to the crossed nociceptive, synkinetic, and tonic neck reflexes were not much help in the differentiation of types of cerebral palsy.

Kenneth Tyler

Electromyographic Studies of the Pathological Stretch Reflex. A Contribution to the Measurement of Spasticity in Cerebral Motor Disorders
J. MENSCH-DECHENE, C. MONFRAIX, and G. TARDIEU. *Revue française d'etudes cliniques et biologiques* Nov., 1958, **3**, 952-959, 3 figs., 5 refs.

The authors, working at the Hôpital de Bicêtre, Paris, have used electromyography to study the increase in muscular tone associated with cerebral motor disorders. They used a technique previously employed in similar research on normal people. A modified electroencephalograph was used to record the potentials picked up by surface electrodes 20 to 25 mm. in diameter placed in pairs, 4 to 10 cm. apart according to age, one pair on the anterior and one pair on the

posterior aspect of the upper arm. Angular movement at the elbow was recorded on the tracing by means of a potentiometer with two arms, one fixed to the upper arm and the other to the forearm so that the axis of rotation coincided with that of the elbow-joint. The resistance of the biceps brachialis muscle to passive stretching was measured with a strain gauge in a number of cases.

Studies of spasticity were carried out on 15 biceps muscles in 13 spastic children, and of rigidity on 4 biceps muscles in 3 patients, including 2 children and one adult with Parkinsonism without tremor, while in addition 7 cases of athetoid hypertonicity were examined. The following characteristics of the three states are defined. (1) In spasticity electrical silence is found at rest, and silence can also be obtained in less than 60 stretchings, provided that these are very slow. Electrical activity is observed with rapid stretching. The speed threshold above which it is impossible to maintain electrical silence can be defined and used to evaluate therapy. (2) In rigidity the electromyograph findings are identical with those observed in spastic muscles. Contrary to previous reports electrical silence occurs both at rest and with very slow stretching, the speed threshold being very low. (3) In athetoid hypertonicity there is often electrical activity at rest. The impulses are irregular and potentials may diminish or disappear during stretching. Electrical silence may occur at times during both rapid and slow stretching. There is no speed threshold.

These findings are compared with previous observations on normal persons, in whom it was found abnormal not to obtain electrical silence in 60 slow or rapid stretchings. The results were also found to be in accordance with those of a previous dynamometric study of cerebral muscle rigidity. The authors claim that both electromyographic and dynamometric tests provide an objective measure of the efficacy of treatment.

Kenneth Tyler

The Development of Motor Control for Walking in One Hundred Cerebral Palsied Children

G. P. SALWAY. *Physical Therapy Review* Nov., 1958, **38**, 749-755, 1 fig., 15 refs.

The author describes the development of motor abilities which lead to walking in children with cerebral palsy. The pattern of development is similar to the normal sequence of motor activities: before a child can sit alone he must first learn to hold his head up; "he must learn to sit alone before he can stand alone, and he must be able to stand alone before he can learn to walk". Of the 100 children (70 boys and 30 girls), 71 were under the age of 10 years. It was found that 39 of the children were able to pass the walking-ability test, 49 could accomplish one or more of the remaining tests successfully, while 12 were unable to perform any of the tests. A majority (59) of the children were spastics, 28 had athetosis, 3 were ataxic, 2 had rigidity, and 8 had both spasticity and rigidity. Although more spastic than athetoid children had basic control of head balance, sitting balance, and creeping, the number who could stand and walk was very nearly the same in both groups. The author suggests that this discrepancy might be explained by the fact that athetoid children have intermittent periods of normal muscle tone in which they are able to experience normal movements.

In treatment the achievement of head balance should be the first objective, training in sitting balance being introduced while the child is learning head balance.

J. MacD. Holmes

The Prognostic Value of the Muscle Biopsy in the "Floppy Infant"

J. G. GREENFIELD, T. CORNMAN, and G. M. SHY. *Brain* Dec., 1958, **81**, 461-484, 15 figs., bibliography.

At the National Institute of Neurological Diseases and Blindness, Bethesda, Maryland, the authors have studied the clinical and pathological findings (as determined from examination of muscle biopsy specimens) in 13 cases presenting a clinical picture of generalized hypotonia in early infancy. They conclude that cases of this type can be divided into four principal groups—(1) infantile spinal muscular atrophy (Werdnig-Hoffmann disease), (2) congenital muscular dystrophy, (3) "central core disease", and (4) "benign congenital hypotonia"—and that muscle biopsy is of great value in de-